PEEP and CPAP

Jargon is a potent cause of professional divisions, and abbreviations are jargon at its worst. The confusion caused by their use is evident in disciplines such as intensive care, in which they have to be understood by many specialists cooperating in the care of patients.

PEEP is positive end expiratory pressure and CPAP is continuous positive airway pressure—two techniques of intensive respiratory care. Both were devised for improving the gaseous exchange in patients with acute disorders of the lung. Their effects on the lungs have to be set against effects on the cardiovascular system—due largely to the net change in intrathoracic pressure they produce.

Alveolar ventilation depends on gas reaching the alveoli, which it can do only if the airways and the alveoli remain patent. Secretions and oedema may cause narrowing, as may increased smooth muscle tone and extrinsic pressure. When alveolar ventilation is improved (less air trapping and better gas mixing) arterial oxygenation improves as a direct consequence.

Ventilation using PEEP depends on fitting a loaded valve, whose setting is independent of gas flow, to the expiratory limb of a ventilator. The effect is that during the expiratory phase of intermittent positive pressure ventilation the airway pressure does not fall to atmospheric pressure. The residual pressure in the airways may be adjusted from conventional levels (5-20 cm water) to as high as 40 cm water.

The raised pressure increases the functional residual capacity of the lungs by increasing the alveolar volume and by alveolar "recruitment"—the opening up of collapsed or poorly ventilated alveoli.[1] The effects of altering the lung volume at which airway closure occurs are disputed. PEEP cannot decrease extravascular lung water—indeed, it increases it.[2-4] The venous return may be grossly impaired,[5] and so the cardiac output may fall; this effect may be reduced by the administration of intravenous fluids.[6] Right ventricular dysfunction may, however, result from the increased overload, and by shifting the interventricular septum leftward may impair left ventricular function, thus further contributing to the low cardiac output.[7] Negatively inotropic humoral agents from the lung may also play some part in the cardiac dysfunction.[8] At high levels of PEEP inotropic support is often required, but it may be ineffective because of the physical distortion of the left ventricle.[7]

CPAP (also known as spontaneous PEEP (sPEEP)) is used in patients breathing spontaneously. It was first introduced by Gregory *et al* in the management of the idiopathic respiratory distress syndrome in the newborn.[9] In these circumstances CPAP was applied either by a tracheal tube or by a plastic pressure chamber, with a snug neck fitting, placed over the baby's head. The pressure of the oxygen enriched air was maintained continuously above atmospheric. Since the pressure was raised throughout the respiratory cycle inspiration was assisted and gaseous exchange improved; oxygenation improved and the respiratory workload decreased. Neonatal mortality from the idiopathic respiratory distress syndrome fell from about 70% to 30%.

CPAP is now used in adults breathing spontaneously using either a mask[10] or a tracheal tube. The use of weighted reservoir bags from which the fresh gas is delivered minimises the problem of falls in pressure during the peak inspiratory flow.[11]

The rise of the intrathoracic pressure that results from either PEEP or CPAP should be rationalised in terms of indications, optimal "dose," and complications. There is some evidence to suggest that the use of PEEP or CPAP reduces the frequency of lung dysfunction in conditions which carry a high risk of the adult respiratory distress syndrome.[12-14] On the other hand, a recent tightly controlled clinical trial showed no benefit from prophylactic PEEP.[15] PEEP overcomes the increase in pulmonary shunting caused by the infusion of dopamine.[16] Its effectiveness during neurosurgery to prevent air embolism is dependent on the intracranial venous pressure, and it appears more effective when the venous pressure is quite negative.[17] The main indication for the use of raised airway pressures, however, remains the need for alveolar recruitment. Expiratory pressure volume curves may show evidence of gross airway closure, demonstrating this need.[18]

There are three "doses" of PEEP: minimal effective, "best," and optimal. Minimal effective PEEP is the pressure at which alveolar recruitment begins.[19] Best PEEP is that end expiratory pressure at which the intrapulmonary shunt fraction of the cardiac output is decreased to less than 15%.[20] This may require the use of very high pressures—more than 40 cm water (super PEEP)—to open up those alveoli resistant to the effects of conventional pressures. Optimal PEEP is the airway pressure at which oxygen transport (cardiac output times arterial oxygen content) is maximal[21]—which implies the best compromise between the pulmonary and the cardiac effects of raised airway pressures. This airway pressure is often the pressure at which pulmonary compliance is maximal.

Apart from the effects on the heart there are few complications of raised airway pressures. Barotrauma, pneumothorax, is no more frequent at higher pressures and seems to be more closely related to the underlying lung disease.[22] During anaesthesia for pulmonary surgery the normal anaesthetic practice is to collapse one lung. If PEEP is applied to the other, dependent lung it does not always improve oxygenation. The pulmonary blood flow may be redirected through the collapsed lung, reducing arterial oxygenation. This effect has been shown to occur in animals with a totally collapsed lobe.[23] What does improve arterial oxygenation during surgery to one lung is to maintain the upper, collapsed lung in a state of partial inflation by insufflating oxygen to a pressure of 10 cm water.[24] Since the lung is neither breathing spontaneously nor being ventilated this should perhaps be termed "constant raised airway pressure."

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Stanley Royd: the epidemiological lesson

The report of the committee of inquiry into an outbreak of food poisoning at Stanley Royd Hospital described the detailed and painstaking investigation carried out by its three members and ended with many welcome recommendations which should help to prevent food poisoning in hospitals in the future.[1] Yet we believe that one vital ingredient was omitted.

As the committee pointed out, the recommendations that prevent the immediate cause of outbreaks of food poisoning —those concerned with faults in the preparation and handling of food—have all been made previously. The problem has been that they are not always put into practice. The tragedy which occurred at Stanley Royd and the monitoring arrangements suggested by the committee should ensure that in future these existing recommendations will be enforced continuously throughout the National Health Service. We commend the committee's recommendations for kitchen inspections, particularly the inclusion in this work of the control of infection officer, who should be a medical consultant, usually a microbiologist. The committee also recommended the appointment of a control of infection nurse in each health district, a long overdue development, and we suggest that there should be at least one such nurse in each district.

The committee also recommended that the Public Health Laboratory Service and its Communicable Disease Surveillance Centre (CDSC) should play a more active part in the control of outbreaks and in training and teaching, and these should be important developments which will help control of infection officers and medical officers for environmental health. Now that the threat to the future of this service by the review of the Department of Health and Social Security has been removed public health laboratories and the CDSC should be able to take on these tasks.

The missing factor in the committee's recommendations concerned the epidemiological aspects of the outbreak. The report failed to identify the need for a more professional approach to epidemiological investigations. The investigation that was undertaken may best be described as "epidemiology by committee": various staff—inexperienced and untrained in epidemiological methods—were given tasks of interviewing affected hospital staff and patients and investigating possible sources of the outbreak without any prior instruction. Such epidemiological deficiencies are sadly not unique: a study reported in the *BMJ* of hospital outbreaks between 1980 and 1982 found that the data necessary to determine the source of infection were often not available.[2] In the Stanley Royd outbreak the acting local medical officer for environmental health should in our view have been responsible to the infectious diseases committee for leading and managing the epidemiological investigation, not simply coordinating it as its chairman. The report criticised the regional medical officer for what appear to be inadequacies of the local epidemiological investigation, for which he had no responsibility.

In its recommendations the committee mentioned the need for local outbreak plans. These may indeed be helpful, but the prior distribution of standardised food history questionnaires is, we believe, of little value and may be misleading because they may not take into account factors important in a particular outbreak, especially as most of those due to salmonellas in hospital are not food borne.[2] Epidemiological data on both cases and controls and clinical data are as vital as the bacteriological findings in determining the source and vehicle of infection.[3] The prime need is for an epidemiologist to take charge of the investigation and control of an outbreak. It is not enough for this local epidemiologist, be it the medical officer for environmental health or the control of infection officer, to "co-ordinate the epidemiological investigation," as the committee recommends. One or other of these medical staff should have specific responsibility for managing the outbreak on behalf of the control of infection committee.

At present the local epidemiologist is usually a community physician appointed as proper officer for infectious disease by the local authority, and in this capacity he or she is described as the medical officer for environmental health.[4] Often such physicians are poorly trained in the investigation of outbreaks, as the committee appreciated. Usually they have no staff available to help them in this work, and sometimes they have many additional duties (as indeed was the case in Wakefield). Furthermore, their responsibility for the prevention of non-notifiable diseases, in the control of infections in hospitals, and in the wider aspects of prevention of non-communicable disease is not clear.

The committee was asked to recommend ways of improving the investigation and control of outbreaks in the future. In our view the most important improvement that is needed